

E#	FILE	FREQUENCY	TERM
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E1	USPAT		BEDLEK, GREGORY J/IN
E2	USPAT	1	BEDLEK, GREGORY J/IN
E3	USPAT	0 -->	BEDNAR/IN
E4	USPAT	1	BEDNAR, ALLAN F/IN
E5	USPAT	2	BEDNAR, BOHUMIL/IN
E6	USPAT	2	BEDNAR, CHARLES/IN
E7	USPAT	3	BEDNAR, CHARLES J/IN
E8	USPAT	1	BEDNAR, DONNA M/IN
E9	USPAT	1	BEDNAR, ERNEST G/IN
E10	USPAT	2	BEDNAR, EUGENE D/IN
E11	USPAT	6	BEDNAR, FRED H/IN
E12	USPAT	7	BEDNAR, GREGORY M/IN
=> e			
E13	USPAT	1	BEDNAR, JAN/IN
E14	USPAT	2	BEDNAR, JIRI/IN
E15	USPAT	1	BEDNAR, JOHN/IN
E16	USPAT	1	BEDNAR, JOHN G/IN
E17	USPAT	6	BEDNAR, JOHN M/IN
E18	USPAT	1	BEDNAR, JOHN P/IN
E19	USPAT	1	BEDNAR, JOHN S/IN
E20	USPAT	1	BEDNAR, JOSEF/IN
E21	USPAT	1	BEDNAR, JOSEPH A/IN
E22	USPAT	1	BEDNAR, JOSEPH A JR/IN
E23	USPAT	1	BEDNAR, LADISLAV/IN
E24	USPAT	1	BEDNAR, MARTIN M/IN
=> e			
E25	USPAT	1	BEDNAR, MILOSLAV/IN
E26	USPAT	1	BEDNAR, REBECCA/IN
E27	USPAT	4	BEDNAR, RICHARD D/IN
E28	USPAT	1	BEDNAR, ROBERT M/IN
E29	USPAT	5	BEDNAR, THOMAS R/IN
E30	USPAT	1	BEDNAR, TINA J/IN
E31	USPAT	1	BEDNAR, VLADIMIR/IN
E32	USPAT	1	BEDNAR, WILLIAM E/IN
E33	USPAT	3	BEDNAR, WILLIAM J/IN
E34	USPAT	1	BEDNARCHUK, JURY VLADIMIROVICH/IN
E35	USPAT	1	BEDNARCYK, WILLIAM S/IN
E36	USPAT	3	BEDNARCZUK, DANIEL/IN

=> s e24

L1 1 "BEDNAR, MARTIN M"/IN

=> d 11 1

1. 5,753,702, May 19, 1998, Arachidonic acid metabolite, 16-hete;
Martin M. Bednar, et al., 514/552 [IMAGE AVAILABLE]

=> s (ischemic or ischemia) (P) (obstruct?)

4491 ISCHEMIC
4419 ISCHEMIA
74453 OBSTRUCT?
L2 517 (ISCHEMIC OR ISCHEMIA) (P) (OBSTRUCT?)

=> s 12 (P) (cd18?)

242 CD18?

L3 0 L2 (P) (CD18?)

=> s 12 and cd18?

242 CD18?
L4 13 L2 AND CD18?

=> d 14 1-13

1. 5,800,815, Sep. 1, 1998, Antibodies to P-selectin and their uses; Robert W. Chestnut, et al., 424/153.1, 133.1, 143.1, 173.1; 435/7.24, 70.21, 326, 328, 343, 346; 530/387.3, 388.2, 388.7, 389.6; 536/23.53 [IMAGE AVAILABLE]
2. 5,767,241, Jun. 16, 1998, Soluble form of GMP-140; Rodger P. McEver, 530/350; 435/69.1, 252.3, 254.11, 325; 530/395; 536/23.5 [IMAGE AVAILABLE]
3. 5,753,617, May 19, 1998, Peptide inhibitors of cellular adhesion; George A. Heavner, et al., 514/9, 15; 530/317, 328 [IMAGE AVAILABLE]
4. 5,728,685, Mar. 17, 1998, Methods of treating inflammation using cell adhesion inhibitors; Saeed A. Abbas, et al., 514/53, 54, 61, 825, 885, 886, 921 [IMAGE AVAILABLE]
5. 5,710,123, Jan. 20, 1998, Peptide inhibitors of selectin binding; George A. Heavner, et al., 514/2, 9, 15; 530/300, 317, 321, 328, 333, 334 [IMAGE AVAILABLE]
6. 5,622,701, Apr. 22, 1997, Cross-reacting monoclonal antibodies specific for E- and P-selectin; Ellen L. Berg, 424/153.1, 143.1, 152.1, 172.1, 173.1; 435/70.21, 334, 343, 451, 452; 530/387.1, 387.3, 388.1, 388.22, 388.7, 389.6; 536/23.53 [IMAGE AVAILABLE]
7. 5,618,785, Apr. 8, 1997, Peptide inhibitors of selectin binding; George A. Heavner, et al., 514/2; 530/328 [IMAGE AVAILABLE]
8. 5,602,230, Feb. 11, 1997, Peptide inhibitors of selectin binding; George A. Heavner, et al., 530/327, 328, 329, 330 [IMAGE AVAILABLE]
9. 5,591,835, Jan. 7, 1997, Substituted lactose derivatives; Saeed A. Abbas, et al., 536/4.1, 123.1, 123.13, 124 [IMAGE AVAILABLE]
10. 5,464,935, Nov. 7, 1995, Peptide inhibitors of selectin binding; George A. Heavner, et al., 530/329, 330 [IMAGE AVAILABLE]
11. 5,378,464, Jan. 3, 1995, Modulation of inflammatory responses by administration of GMP-140 or antibody to GMP-140; Rodger P. McEver, 424/143.1; 514/8 [IMAGE AVAILABLE]
12. 5,198,424, Mar. 30, 1993, Functionally active selectin-derived peptides; Rodger P. McEver, 514/13; 424/1.37, 1.69; 427/2.24, 2.25; 514/12, 14, 15, 16; 530/324, 325, 326, 327; 623/11 [IMAGE AVAILABLE]
13. 4,797,277, Jan. 10, 1989, Method for reperfusion therapy; Karl E. Arfors, 435/1.2; 424/143.1, 153.1; 514/2, 21; 530/388.7 [IMAGE AVAILABLE]

=> d 14 1-13 kwi

'KWI' IS NOT A VALID FORMAT FOR FILE 'USPAT'
ENTER DISPLAY FORMAT (CIT):kwic

US PAT NO: 5,800,815 [IMAGE AVAILABLE]

L4: 1 of 13

DETDESC:

DETD(90)

Ischemia/reperfusion injury is an inflammatory condition that occurs on restoring blood flow to organs suffering from an **obstructed** supply causing **ischemia** (oxygen deprivation). Unless rapidly relieved by reperfusion, **ischemia** causes death of surrounding cells, and eventually, death of a whole organ or patient. However, accumulating evidence suggests that reperfusion. . . . least in part from an inflammatory response mediated by activated neutrophils in the restored blood flow. Some patients have whole-body **ischemia**, whereas in other patients **ischemia** is confined to particular parts or organs of the body. For example, a patient may suffer from epidermal, myocardial, renal, cerebral, splenic, hepatic, spinal, splanchnic, pulmonary, partial-body, or whole-body **ischemia**. The therapeutic agents of the invention function by antagonizing the interaction of such lymphocytes with P-selectin.

DETDESC:

DETD(93)

Therapeutic . . . other molecules, particularly humanized or human antibodies reactive with different adhesion molecules. Suitable immunoglobulins include those specific for CD11a, CD11b, **CD18**, E-selectin, L-selectin and ICAM-1. The immunoglobulins should bind to epitopes of these adhesion molecules so as to inhibit binding of. . . .

US PAT NO: 5,767,241 [IMAGE AVAILABLE]

L4: 2 of 13

SUMMARY:

BSUM(3)

The . . . step in migration of leukocytes to tissues in response to microbial invasion. Although a class of inducible leukocyte receptors, the CD11-**CD18** molecules, are thought to have some role in adherence to endothelium, mechanisms of equal or even greater importance for leukocyte. . . .

SUMMARY:

BSUM(6)

The . . . can be involved in both inflammatory and coagulation processes. For example, the Mac-1 receptor on leukocytes, a member of the CD11-**CD18** group, mediates phagocytosis and serves as a receptor for the degradation product of complement C3bi, is involved in one pathway. . . .

DETDESC:

DETD(94)

Reperfusion injury is a major problem in clinical cardiology. Therapeutic agents that reduce leukocyte adherence in **ischemic** myocardium can significantly enhance the therapeutic efficacy of thrombolytic agents. Thrombolytic therapy with agents such as tissue plasminogen activator or streptokinase can relieve coronary artery **obstruction** in many patients with severe myocardial **ischemia** prior to irreversible myocardial cell death. However, many such patients still suffer myocardial neurosis despite restoration of blood flow. This "reperfusion injury" is known to be associated with adherence of

leukocytes to vascular endothelium in the **ischemic** zone, presumably in part because of activation of platelets and endothelium by thrombin and cytokines that makes them adhesive for leukocytes (Romson et al., Circulation 67: 1016-1023, 1983). These adherent leukocytes can migrate through the endothelium and destroy **ischemic** myocardium just as it is being rescued by restoration of blood flow.

US PAT NO: 5,753,617 [IMAGE AVAILABLE]

L4: 3 of 13

SUMMARY:

BSUM(4)

Leukocyte . . . initial step in migration of leukocytes to tissues in response to microbial invasion. A class of inducible leukocyte receptors, the CD11-**CD18** molecules (integrins), have a role in adherence to endothelium. These molecules are involved in mechanisms of leukocyte adherence involving inducible. . .

DETDESC:

DETD(42)

Reperfusion injury is a major problem in clinical cardiology. Therapeutic agents that reduce leukocyte adherence in **ischemic** myocardium can significantly enhance the therapeutic efficacy of thrombolytic agents. Thrombolytic therapy with agents such as tissue plasminogen activator or streptokinase can relieve coronary artery **obstruction** in many patients with severe myocardial **ischemia** prior to irreversible myocardial cell death. However, many such patients still suffer myocardial neurosis despite restoration of blood flow. This "reperfusion injury" is known to be associated with adherence of leukocytes to vascular endothelium in the **ischemic** zone, presumably in part because of activation of platelets and endothelium by thrombin and cytokines that makes them adhesive for leukocytes (Romson et al., Circulation 67: 1016-1023 (1983)). These adherent leukocytes can migrate through the endothelium and destroy **ischemic** myocardium just as it is being rescued by restoration of blood flow.

US PAT NO: 5,728,685 [IMAGE AVAILABLE]

L4: 4 of 13

DETDESC:

DETD(30)

Reperfusion injury is a major problem in clinical cardiology. Therapeutic agents that reduce leukocyte adherence in **ischemic** myocardium can significantly enhance the therapeutic efficacy of thrombolytic agents. Thrombolytic therapy with agents such as tissue plasminogen activator or streptokinase can relieve coronary artery **obstruction** in many patients with severe myocardial **ischemia** prior to irreversible myocardial cell death. However, many such patients still suffer myocardial neurosis despite restoration of blood flow. This "reperfusion injury" is known to be associated with adherence of leukocytes to vascular endothelium in the **ischemic** zone, presumably in part because of activation of platelets and endothelium by thrombin and cytokines that makes them adhesive for leukocytes (Romson et al., Circulation 67:1016-1023, 1983). These adherent leukocytes can migrate through the endothelium and **ischemic** myocardium just as it is being rescued by restoration of blood flow.

DETDESC:

DETD(185)

It . . . inhibition seen using pharmacological agents, including a number of peptides derived from P-selectin and antibodies directed against P-selectin and the **CD11b/CD18** complex (Ma, Xin-liang, et al., Circulation (1993) 88-2:649), has been 40%. Compound 13b provides a degree of inhibition similar to. . .

US PAT NO: 5,710,123 [IMAGE AVAILABLE]

L4: 5 of 13

SUMMARY:

BSUM(5)

Leukocyte . . . step in migration of leukocytes to tissues in response to microbial invasion. Although a class of inducible leukocyte receptors, the **CD11-CD18** molecules, are thought to have some role in adherence to endothelium, mechanisms of equal or even greater importance for leukocyte. . .

SUMMARY:

BSUM(105)

Reperfusion injury is a major problem in clinical cardiology. Therapeutic agents that reduce leukocyte adherence in **ischemic** myocardium can significantly enhance the therapeutic efficacy of thrombolytic agents. Thrombolytic therapy with agents such as tissue plasminogen activator or streptokinase can relieve coronary artery **obstruction** in many patients with severe myocardial **ischemia** prior to irreversible myocardial cell death. However, many such patients still suffer myocardial neurosis despite restoration of blood flow. This "reperfusion injury" is known to be associated with adherence of leukocytes to vascular endothelium in the **ischemic** zone, presumably in part because of activation of platelets and endothelium by thrombin and cytokines that makes them adhesive for leukocytes (Romson et al., Circulation 67: 1016-1023 (1983)). These adherent leukocytes can migrate through the endothelium and destroy **ischemic** myocardium just as it is being rescued by restoration of blood flow.

US PAT NO: 5,622,701 [IMAGE AVAILABLE]

L4: 6 of 13

SUMMARY:

BSUM(4)

P-selectin . . . (1993); Jones et al., Biophys. J. 65: 1560-1569 (1993); Mayadas et al., Cell 74: 541-554 (1993)). This initial interaction precedes **CD18**-integrin-mediated adhesion and subsequent migration of neutrophils through the endothelium and into inflamed tissue sites (Lawrence et al., Cell 65: 859-873. . .

DETDESC:

DETD(78)

The . . . with other antibodies, particularly antibodies reactive with different adhesion molecules. For example, suitable antibodies include those specific for **CD11a**, **CD11b**, **CD18**, **L-selectin**, and **ICAM-1**. Other suitable antibodies are those specific for lymphokines, such as **IL-1**, **IL-2** and **IFN-.gamma.**, and their receptors.. .

DETDESC:

DETD(79)

In some therapeutic methods of **ischemia**-reperfusion therapy,

crossreacting antibodies are used in combination with thrombolytic agents. In previous methods patients with myocardial infarction or unstable angina are often treated by opening the occluded coronary artery. Reopening of the **obstructed** coronary artery can be achieved by administration of thrombolytic agents which lyse the clot causing the **obstruction**, and which, thereby, restore coronary blood flow. Reperfusion of the vessel can also be achieved by percutaneous transluminal coronary angioplasty (PTCA) by means of balloon dilation of the **obstructed** and narrowed segment of the coronary artery. However, restoration of coronary blood flow leads to **ischemia**-reperfusion injury in prior methods.

US PAT NO: 5,618,785 [IMAGE AVAILABLE]

L4: 7 of 13

SUMMARY:

BSUM(5)

Leukocyte . . . step in migration of leukocytes to tissues in response to microbial invasion. Although a class of inducible leukocyte receptors, the CD11-**CD18** molecules, are thought to have some role in adherence to endothelium, mechanisms of equal or even greater importance for leukocyte. . .

DETDESC:

DETD(60)

Reperfusion injury is a major problem in clinical cardiology. Therapeutic agents that reduce leukocyte adherence in **ischemic** myocardium can significantly enhance the therapeutic efficacy of thrombolytic agents. Thrombolytic therapy with agents such as tissue plasminogen activator or streptokinase can relieve coronary artery **obstruction** in many patients with severe myocardial **ischemia** prior to irreversible myocardial cell death. However, many such patients still suffer myocardial neurosis despite restoration of blood flow. This "reperfusion injury" is known to be associated with adherence of leukocytes to vascular endothelium in the **ischemic** zone, presumably in part because of activation of platelets and endothelium by thrombin and cytokines that makes them adhesive for leukocytes (Romson et al., Circulation 67: 1016-1023 (1983)). These adherent leukocytes can migrate through the endothelium and destroy **ischemic** myocardium just as it is being rescued by restoration of blood flow.

US PAT NO: 5,602,230 [IMAGE AVAILABLE]

L4: 8 of 13

SUMMARY:

BSUM(5)

Leukocyte . . . step in migration of leukocytes to tissues in response to microbial invasion. Although a class of inducible leukocyte receptors, the CD11-**CD18** molecules, are thought to have some role in adherence to endothelium, mechanisms of equal or even greater importance for leukocyte. . .

DETDESC:

DETD(57)

Reperfusion injury is a major problem in clinical cardiology. Therapeutic agents that reduce leukocyte adherence in **ischemic** myocardium can significantly enhance the therapeutic efficacy of thrombolytic agents. Thrombolytic therapy with agents such as tissue plasminogen activator or streptokinase can relieve coronary artery

obstruction in many patients with severe myocardial **ischemia** prior to irreversible myocardial cell death. However, many such patients still suffer myocardial neurosis despite restoration of blood flow. This "reperfusion injury" is known to be associated with adherence of leukocytes to vascular endothelium in the **ischemic** zone, presumably in part because of activation of platelets and endothelium by thrombin and cytokines that makes them adhesive for leukocytes (Romson et al., Circulation 67:1016-1023 (1983)). These adherent leukocytes can migrate through the endothelium and destroy **ischemic** myocardium just as it is being rescued by restoration of blood flow.

US PAT NO: 5,591,835 [IMAGE AVAILABLE]

L4: 9 of 13

DETDESC:

DETD(30)

Reperfusion injury is a major problem in clinical cardiology. Therapeutic agents that reduce leukocyte adherence in **ischemic** myocardium can significantly enhance the therapeutic efficacy of thrombolytic agents. Thrombolytic therapy with agents such as tissue plasminogen activator or streptokinase can relieve coronary artery **obstruction** in many patients with severe myocardial **ischemia** prior to irreversible myocardial cell death. However, many such patients still suffer myocardial neurosis despite restoration of blood flow. This "reperfusion injury" is known to be associated with adherence of leukocytes to vascular endothelium in the **ischemic** zone, presumably in part because of activation of platelets and endothelium by thrombin and cytokines that makes them adhesive for leukocytes (Romson et al., Circulation 67:1016-1023, 1983). These adherent leukocytes can migrate through the endothelium and **ischemic** myocardium just as it is being rescued by restoration of blood flow.

DETDESC:

DETD(185)

It . . . inhibition seen using pharmacological agents, including a number of peptides derived from P-selectin and antibodies directed against P-selectin and the CD11b/CD18 complex (Ma, Xin-liang, et al., Circulation (1993) 88-2:649), has been 40%. Compound 13b provides a degree of inhibition similar to. . .

US PAT NO: 5,464,935 [IMAGE AVAILABLE]

L4: 10 of 13

SUMMARY:

BSUM(5)

Leukocyte . . . step in migration of leukocytes to tissues in response to microbial invasion. Although a class of inducible leukocyte receptors, the CD11-CD18 molecules, are thought to have some role in adherence to endothelium, mechanisms of equal or even greater importance for leukocyte. . .

DETDESC:

DETD(49)

Reperfusion injury is a major problem in clinical cardiology. Therapeutic agents that reduce leukocyte adherence in **ischemic** myocardium can significantly enhance the therapeutic efficacy of thrombolytic agents. Thrombolytic therapy with agents such as tissue plasminogen activator or streptokinase can relieve coronary artery **obstruction** in many patients with severe myocardial **ischemia**

prior to irreversible myocardial cell death. However, many such patients still suffer myocardial neurosis despite restoration of blood flow. This "reperfusion injury" is known to be associated with adherence of leukocytes to vascular endothelium in the **ischemic** zone, presumably in part because of activation of platelets and endothelium by thrombin and cytokines that makes them adhesive for leukocytes (Romson et al., Circulation 67:1016-1023 (1983)). These adherent leukocytes can migrate through the endothelium and destroy **ischemic** myocardium just as it is being rescued by restoration of blood flow.

US PAT NO: 5,378,464 [IMAGE AVAILABLE]

L4: 11 of 13

SUMMARY:

BSUM(4)

The . . . step in migration of leukocytes to tissues in response to microbial invasion. Although a class of inducible leukocyte receptors, the CD11-**CD18** molecules, are thought to have some role in adherence to endothelium, mechanisms of equal or even greater importance for leukocyte. . . .

SUMMARY:

BSUM(7)

The . . . can be involved in both inflammatory and coagulation processes. For example, the Mac-1 receptor on leukocytes, a member of the CD11-**CD18** group, mediates phagocytosis and serves as a receptor for the degradation product of complement C3bi, is involved in one pathway.

DETDESC:

DETD(92)

Reperfusion injury is a major problem in clinical cardiology. Therapeutic agents that reduce leukocyte adherence in **ischemic** myocardium can significantly enhance the therapeutic efficacy of thrombolytic agents. Thrombolytic therapy with agents such as tissue plasminogen activator or streptokinase can relieve coronary artery **obstruction** in many patients with severe myocardial **ischemia** prior to irreversible myocardial cell death. However, many such patients still suffer myocardial neurosis despite restoration of blood flow. This "reperfusion injury" is known to be associated with adherence of leukocytes to vascular endothelium in the **ischemic** zone, presumably in part because of activation of platelets and endothelium by thrombin and cytokines that makes them adhesive for leukocytes (Romson et al., Circulation 67: 1016-1023, 1983). These adherent leukocytes can migrate through the endothelium and destroy **ischemic** myocardium just as it is being rescued by restoration of blood flow.

US PAT NO: 5,198,424 [IMAGE AVAILABLE]

L4: 12 of 13

SUMMARY:

BSUM(5)

Leukocyte . . . step in migration of leukocytes to tissues in response to microbial invasion. Although a class of inducible leukocyte receptors, the CD11-**CD18** molecules, are thought to have some role in adherence to endothelium, mechanisms of equal or even greater importance for leukocyte. . . .

DETDESC:

DETD(73)

Reperfusion injury is a major problem in clinical cardiology. Therapeutic agents that reduce leukocyte adherence in **ischemic** myocardium can significantly enhance the therapeutic efficacy of thrombolytic agents. Thrombolytic therapy with agents such as tissue plasminogen activator or streptokinase can relieve coronary artery **obstruction** in many patients with severe myocardial **ischemia** prior to irreversible myocardial cell death. However, many such patients still suffer myocardial neurosis despite restoration of blood flow. This "reperfusion injury" is known to be associated with adherence of leukocytes to vascular endothelium in the **ischemic** zone, presumably in part because of activation of platelets and endothelium by thrombin and cytokines that makes them adhesive for leukocytes (Romson et al., Circulation 67: 1016-1023, 1983). These adherent leukocytes can migrate through the endothelium and destroy **ischemic** myocardium just as it is being rescued by restoration of blood flow.

US PAT NO: 4,797,277 [IMAGE AVAILABLE]

L4: 13 of 13

SUMMARY:

BSUM(3)

Ischemia is a condition that occurs in organs suffering from an **obstructed** blood flow. **Ischemic** conditions that are not rapidly abolished may lead to cell death and may be fatal for the organ or individual involved. Since quite a long time it has been recognized that the reperfusion of an organ suffering from **ischemia** may lead to I/R-induced tissue damage in a variety of clinical conditions like coronary infarction, organ transplantation, shock etc.

SUMMARY:

BSUM(11)

LACs . . . and monocytes, Mac-1 on granulocytes and monocytes, and p150.95 on macrophages and monocytes. Each subunit consists of one common beta-chain (**CD18**) and an alpha-chain that is unique for each of the three subunits (CD11a, CD11b, and CD11c, respectively). The LAC complex.

DETDDESC:

DETD(9)

The . . . of the invention may be part of a prepacked kit containing therapeutics useful for the treatment of different aspects of **ischemia**/reperfusion, so-called multi-factorial treatment. As has been indicated above reperfusion damage can appear when an **obstructed** blood-flow is cleared giving as a consequence the release of ROM, cationic proteins and proteases. Thus therapeutically active free radical. . .

DETDDESC:

DETD(26)

A monoclonal antibody (MoAb 60.3) to LAC (**CD18**) was tested for its effect on adherence of feline neutrophils using the method of Fehr and Dahinden. Briefly, one ml. . .

=> d his

(FILE 'USPAT' ENTERED AT 16:42:19 ON 29 OCT 1998)
E BEDNAR/IN
L1 1 S E24
L2 517 S (ISCHEMIC OR ISCHEMIA) (P) (OBSTRUCT?)
L3 0 S L2 (P) (CD18?)
L4 13 S L2 AND CD18?

=> d 14 1-17 date

13 ANSWERS ARE AVAILABLE. SPECIFIED ANSWER NUMBER EXCEEDS ANSWER SET
SIZE
ENTER ANSWER NUMBER OR RANGE (1):1-13

L4: 1 of 13
TITLE: Antibodies to P-selectin and their uses
US PAT NO: 5,800,815 DATE ISSUED: Sep. 1, 1998
[IMAGE AVAILABLE]
APPL-NO: 08/202,047 DATE FILED: Feb. 25, 1994
FRN-PR. NO: 105614 FRN FILED: May 5, 1993
FRN-PR. CO: Israel
FRN-PR. NO: PCT/US93/04274 FRN FILED: May 4, 1993
FRN-PR. CO: World Intellectual Property Organization
REL-US-DATA: Continuation-in-part of Ser. No. 57,292, May 5, 1993,
abandoned, which is a continuation-in-part of Ser. No.
880,198, May 5, 1992, abandoned.

L4: 2 of 13
TITLE: Soluble form of GMP-140
US PAT NO: 5,767,241 DATE ISSUED: Jun. 16, 1998
[IMAGE AVAILABLE]
APPL-NO: 08/272,224 DATE FILED: Jul. 8, 1994
REL-US-DATA: Continuation of Ser. No. 320,408, Mar. 8, 1989, Pat. No.
5,378,464.

L4: 3 of 13
TITLE: Peptide inhibitors of cellular adhesion
US PAT NO: 5,753,617 DATE ISSUED: May 19, 1998
[IMAGE AVAILABLE]
APPL-NO: 08/397,101 DATE FILED: Mar. 7, 1995
PCT-NO: PCT/US93/08504 PCT-FILED: Sep. 8, 1993
371-DATE: Mar. 7, 1995
102(E)-DATE: Mar. 7, 1995
PCT-PUB-NO: WO94/05310 PCT-PUB-DATE: Mar. 17, 1994
REL-US-DATA: Continuation-in-part of Ser. No. 941,653, Sep. 8, 1992,
abandoned.

L4: 4 of 13
TITLE: Methods of treating inflammation using cell adhesion
inhibitors
US PAT NO: 5,728,685 DATE ISSUED: Mar. 17, 1998
[IMAGE AVAILABLE]
APPL-NO: 08/466,667 DATE FILED: Jun. 6, 1995
REL-US-DATA: Division of Ser. No. 189,630, Feb. 1, 1994, Pat. No.
5,591,835, which is a continuation-in-part of Ser. No.

TITLE: Peptide inhibitors of selectin binding L4: 5 of 13
 US PAT NO: 5,710,123 DATE ISSUED: Jan. 20, 1998
 [IMAGE AVAILABLE]
 APPL-NO: 08/454,207 DATE FILED: Jun. 9, 1995
 PCT-NO: PCT/US93/12110 PCT-FILED: Dec. 13, 1993
 371-DATE: Jun. 9, 1995
 102(E)-DATE: Jun. 9, 1995
 PCT-PUB-NO: WO94/14836 PCT-PUB-DATE: Jul. 7, 1994
 REL-US-DATA: Continuation-in-part of Ser. No. 997,771, Dec. 18, 1992,
 abandoned.

TITLE: Cross-reacting monoclonal antibodies specific for E- and L4: 6 of 13
 P-selectin
 US PAT NO: 5,622,701 DATE ISSUED: Apr. 22, 1997
 [IMAGE AVAILABLE]
 APPL-NO: 08/259,963 DATE FILED: Jun. 14, 1994

TITLE: Peptide inhibitors of selectin binding L4: 7 of 13
 US PAT NO: 5,618,785 DATE ISSUED: Apr. 8, 1997
 [IMAGE AVAILABLE]
 APPL-NO: 08/457,804 DATE FILED: Jun. 1, 1995
 REL-US-DATA: Continuation of Ser. No. 156,415, Nov. 22, 1993,
 abandoned.

TITLE: Peptide inhibitors of selectin binding L4: 8 of 13
 US PAT NO: 5,602,230 DATE ISSUED: Feb. 11, 1997
 [IMAGE AVAILABLE]
 APPL-NO: 08/438,475 DATE FILED: May 10, 1995
 REL-US-DATA: Continuation of Ser. No. 889,650, May 19, 1992, abandoned.

TITLE: Substituted lactose derivatives L4: 9 of 13
 US PAT NO: 5,591,835 DATE ISSUED: Jan. 7, 1997
 [IMAGE AVAILABLE]
 APPL-NO: 08/189,630 DATE FILED: Feb. 1, 1994
 REL-US-DATA: Continuation-in-part of Ser. No. 910,709, Jun. 29, 1992,
 abandoned.

TITLE: Peptide inhibitors of selectin binding L4: 10 of 13
 US PAT NO: 5,464,935 DATE ISSUED: Nov. 7, 1995
 [IMAGE AVAILABLE]
 APPL-NO: 08/384,680 DATE FILED: Feb. 6, 1995
 REL-US-DATA: Continuation of Ser. No. 891,986, May 28, 1992, abandoned.

TITLE: Modulation of inflammatory responses by administration of L4: 11 of 13
 GMP-140 or antibody to GMP-140
 US PAT NO: 5,378,464 DATE ISSUED: Jan. 3, 1995
 [IMAGE AVAILABLE]
 APPL-NO: 07/320,408 DATE FILED: Mar. 8, 1989

TITLE: Functionally active selectin-derived peptides L4: 12 of 13
 US PAT NO: 5,198,424 DATE ISSUED: Mar. 30, 1993
 [IMAGE AVAILABLE]
 APPL-NO: 07/867,271 DATE FILED: Apr. 7, 1992
 REL-US-DATA: Continuation of Ser. No. 554,199, Jul. 17, 1990,
 abandoned, which is a continuation-in-part of Ser. No.

320,408, Mar. 8, 1989.

TITLE:
US PAT NO:

Method for reperfusion therapy

4,797,277

[IMAGE AVAILABLE]

APPL-NO:

07/099,403

DATE ISSUED:

L4: 11 of 13

Jan. 10, 1989

DATE FILED:

Sep. 22, 1987

3/7/1 (Item 1 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)
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11663442 BIOSIS Number: 98263442

A P-selectin-immunoglobulin G chimera is protective in a rabbit ear model of **ischemia**-reperfusion

Lee W P; Gribling P; De Guzman L; Ehsani N; Watson S R

MS 32, Dep. Immunol., Genentech, 460, Pt. San Bruno Blvd., South San Francisco, CA 94080, USA

Surgery (St Louis) 117 (4). 1995. 458-465.

Full Journal Title: Surgery (St Louis)

ISSN: 0039-6060

Language: ENGLISH

Print Number: Biological Abstracts Vol. 099 Iss. 012 Ref. 168360

Background: Neutrophils have been shown to play a role in **ischemia**-reperfusion injury, and the initial interaction of neutrophils with the endothelium is mediated through the selectin family of adhesion molecules. Thus the purpose of these studies was to determine whether a P-selectin-IgG chimera was protective in a model of **ischemia**-reperfusion injury.

Methods: The model used was a rabbit ear model of **ischemia**-reperfusion. Selectin-IgG chimeras were given at the time of reperfusion of the tissue, and their efficacy was compared with an anti-CD18 antibody (**MHM23**). Results: The P-selectin-IgG was as protective in this model as an anti-CD18 antibody. The chimera did not mediate its effect by causing the animals to become neutropenic. Conclusions: P-selectin plays a role in **ischemia** -reperfusion injury. This is in agreement with data from other groups. The fact that the chimera was effective in this model suggests that carbohydrates or small molecule mimics of carbohydrates would be effective in this model. Such antiinflammatory agents may have fewer side effects in terms of increased risk of sepsis.

008503079 **Image available**

WPI Acc.No: 91-007163/199101

New hybridoma cell lines and monoclonal antibodies - reactive with
leukocyte adhesion receptor beta-chain, for treatment of immune
response-mediated disorders, e.g. AIDS

Patent Assignee: UNIV. JOHNS HOPKINS SCHOOL MED. (UYJO); UNIV. JOHNS HOPKINS
(UYJO); UNIV. JOHNS HOPKINS SCHOOL MEDICINE, (UYJO)

Inventor: HILDRETH J E

Number of Countries: 016 Number of Patents: 012

Patent Family:

Patent No	Kind	Date	Applicat No	Kind	Date	Main IPC	Week
WO 9015076	A	19901213					199101 B
AU 9058471	A	19910107					199115
EP 432249	A	19910619	EP 90909864	A	19900530		199125
JP 4501365	W	19920312	JP 90509217	A	19900530		199217
AU 645016	B	19940106	AU 9058471	A	19900530	C12P-021/08	199408
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			AU 9350353	A	19931028		
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			WO 90US2979	A	19900530		
DE 69028684	E	19961031	DE 628684	A	19900530	C07K-016/28	199649
			EP 90909864	A	19900530		
			WO 90US2979	A	19900530		
ES 2095876	T3	19970301	EP 90909864	A	19900530	C07K-016/28	199716

Priority Applications (No Type Date): US 89361271 A 19890602

Cited Patents: NoSR.Pub; 3. journal ref.; EP 314863; EP 346078; EP 365837;
US 4506009; WO 8806592

Patent Details:

Patent	Kind	Lan	Pg	Filing Notes	Application	Patent
WO 9015076	A					

Designated States (National): AU CA JP

Designated States (Regional): AT BE CH DE DK ES FR GB IT LU NL SE

EP 432249 A
 Designated States (Regional): AT BE CH DE ES FR GB IT LI LU NL SE
 JP 4501365 W
 AU 645016 B Previous Publ. AU 9058471
 Based on WO 9015076
 AU 9350353 A Div. ex. AU 9058471
 AU 666977 B Div. ex. AU 9058471
 Previous Publ. AU 9350353
 EP 432249 B1 E 14 Based on WO 9015076
 Designated States (Regional): AT BE CH DE DK ES FR GB IT LI LU NL SE
 DE 69028684 E Based on EP 432249
 Based on WO 9015076
 ES 2095876 T3 Based on EP 432249

Abstract (Basic): WO 9015076 A

New continuous hybridoma cell lines are capable of secreting mAbs reactive with leukocyte adhesion receptor beta-chain and suppressing intercellular leukocyte adhesion. Pref. the receptor is one of LFA-1, Mac-1 and Leu M5. The hybridoma is pref. ATCC HBX or its isotype switch variants. The mAbs are also claimed.

Immune response-mediated disorders are ameliorated by admin. of the new Ab. Specific disorders treated and AIDS, autoimmune disease and graft rejection. Admin. is pref. parenteral, esp. by subcutaneous, intramuscular, intraperitoneal, intracavity, transdermal or i.v. injection. Dose is pref. 0.01-2000 mg/kg/dose. The mAb may be therapeutically labelled, pref. by a radioisotope, drug, lectin or toxin.

Leukocyte adhesion receptor is determined by contacting a suspected source with the labelled mAb or fragment having the specificity of mAb H52 and its isotype switch variants and determining whether the mAb binds to the source. Detection is pref. in vivo using a radioisotope or paramagnetic label or in vitro using a radioisotope, fluorescent compound, colloidal metal, chemiluminescent compound, bioluminescent cpd. or an enzyme.

USE/ADVANTAGE -- The mAbs suppress the ability of the leukocytes to adhere to each other, thus decreasing the likelihood of cell-to-cell transmission of infectious agents and immune response activation.

Dwg. 2/4

Abstract (Equivalent): EP 432249 B
 A continuous hybridoma cell line which secretes monoclonal antibodies with the binding specificity of the H52 monoclonal antibody which is secreted by hybridoma cell line ATCC HB 10160

(Dwg. 0/4)

Derwent Class.: B04; D16; S03

International Patent Class (Main): C07K-016/28; C12P-021/08

International Patent Class (Additional): A61K-039/39; A61K-039/395;

A61K-043/00; A61K-051/00; C07K-015/28; C12N-005/12; G01N-033/53;

G01N-033/535; G01N-033/577; G01N-033/68